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Observations on a New Anthelmintic (Bephenium Embonate): Its Use Against Nematodirus in Lambs

BY

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SUMMARY.—A new anthelmintic, bephenium embonate, effective against larval and adult stages of Nematodirus is described.

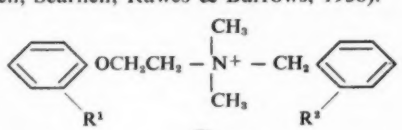
Details are given of the methods used and of the results obtained in assessing its efficiency.

A dosing routine likely to be effective for prophylaxis is suggested.

Introduction

THERE have been several reports of outbreaks in Britain of the disease of young lambs caused by heavy burdens of *Nematodirus battus* and *N. filicollis* (Crofton & Thomas, 1951; Kingsbury, 1953; Heath, 1955; Stamp, Dunn & Watt, 1955; Thomas & Stevens, 1956; Baxter, 1957). The disease causes severe losses particularly in certain years.

Members of the series of drugs of the general formula (I) have been shown to have marked activity against a number of nematodes including several of those causing parasitic gastro-enteritis in sheep (Copp, Standen, Scarnell, Rawes & Burrows, 1958).



(I)

The first drug of the series to be tested against Nematodirus was di-(*N*-benzyl-*N* : *N*-dimethyl-*N*-2-phenoxyethylammonium) embonate (monohydrate), formula (II), which has been given the open name of bephenium embonate.* It is a pale yellow solid, with a melting point of 153° to 156° C. and a solubility in water at 20° C. of 0.04 per cent. It has no observed staining properties. (See formula (II) overleaf.)

This paper records our observations on the anthelmintic efficiency of bephenium embonate against Nematodirus in lambs.

Method

The first observations on the effect of this drug on Nematodirus were made in 1956 during the course of general anthelmintic tests. Later, a small group of lambs passing moderately high numbers of Nematodirus eggs in the faeces was selected for test purposes.

More extensive observations were not possible until the spring of 1957, when field trials were carried out on naturally-infested lambs.

Faecal egg counts were made using a modified McMaster technique and worm counts by the usual dilution method.

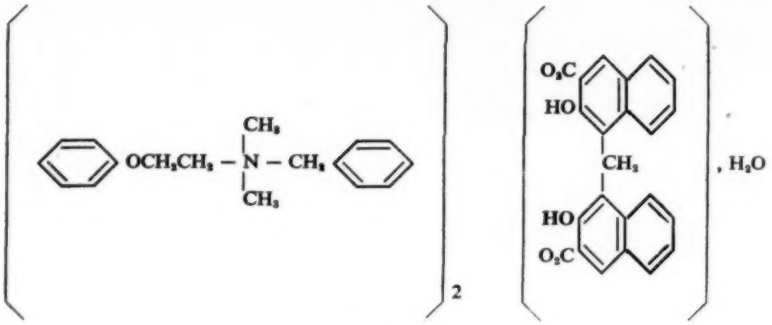
In the initial screening tests bephenium embonate was used in the form of a finely-milled powder, administered *per os* in gelatine capsules. For the field trials, compressed products each containing 2.5 g. of the drug were used, and the dose calculated to the nearest whole product.

Acute toxicity tests were completed on lambs before routine screening for efficiency was started, and later the effect of the drug on weight gains was determined using lambs reared worm-free apart from *Strongyloides papillosus*.

Results

The lambs used in the general anthelmintic screening tests were randomised on the basis of high faecal egg counts of trichostrongyles other than Nematodirus, the counts of which were low. There was, however,

* Issued as a dispersible powder, "Frantin" (B. W. & Co.).



(II)

a clear indication that bephenium embonate was active against this parasite.

Six lambs passing significant numbers of Nematodirus eggs then became available. Bephenium embonate was administered to 2 animals at 1,000 mg. per kg. bodyweight, to 2 at 500 mg. per kg., and 2 were maintained as untreated controls. The faecal counts before and after treatment and the worm counts of animals at slaughter showed marked clearance of Nematodirus from the treated animals and are recorded in Table I.

The first field trial was carried out in lambs in a flock in which infestation with Nematodirus spp. had been present in the previous year, confirmed by faecal egg counts greater than 1,000 e.p.g. in several of the lambs. During this trial, however, infestation was slight, and egg counts remained low. A few tests were carried out using bephenium embonate at the 500, 250 and 125 mg. per kg. dose levels. No Nematodirus eggs were found in faeces samples from lambs after treatment, whereas the egg counts of control untreated lambs were maintained for some time at their previous levels.

The second field trial was carried out in lambs in another flock suffering from clinical Nematodiriasis. A *post-mortem* examination of one of the lambs by a Veterinary Investigation Officer revealed the presence of 20,000 to 25,000 Nematodirus. Before faeces sampling, the 165 available lambs were randomised between a control untreated group and 2 treated groups to receive doses of bephenium embonate of 250 and 125 mg. per kg. respectively. Five days after dosing, the faecal Nematodirus egg counts of lambs dosed with 250 mg. per kg. were dramatically reduced; those dosed with 125 mg. per kg. were markedly reduced; whereas the egg counts of the majority of the untreated controls had increased. The details are set out in Table II.

Further faeces samples were collected 19 days after dosing. The egg counts of those from the control lambs were by this time reduced but were not as low as those of the treated lambs.

Three lambs from each of the treated and control groups were slaughtered 8 days after dosing. The numbers of adult worms in both dosed groups were significantly lower than those in the control group. In addition, the activity of the 250-mg. per kg. dose against the larval stages was clearly demonstrated. The results are shown in Table III. (Page 254.)

The third field trial was carried out in lambs in a flock suffering from parasitic gastro-enteritis apparently due to mixed infestation with Ostertagia and *N. filicollis*. Before the first visit one lamb died and a *post-mortem* examination by a Veterinary Investigation Officer revealed 12,000 Ostertagia and 22,000 Nematodirus. Prior to faeces sampling, the lambs were randomised between a control untreated group and 2 groups to receive bephenium embonate at dose

TABLE I

DATA RELATING TO NEMATODIRUS IN LAMBS SELECTED ON THE BASIS OF FAECAL EGG COUNTS FOR TESTING BEPHENIUM EMBONATE

Lamb number	Eggs per gramme of faeces		Dose (mg. per kg.) administered 3.10.56	Eggs per gramme of faeces		Number of adult Nematodirus at autopsy, 10.10.56
	27.9.56	3.10.56		5.10.56	8.10.56	
H.9	1,300	N.S.*	1,000	N.S.	0	Not killed
H.119	700	800		0	0	Not killed
H.103	2,850	2,850	500	50	0	0
F.47	700	350		0	0	0
H.60	600	350	Nil	N.S.	N.S.	7,560
H.98	600	650		500	450	5,256

* N.S. : No sample obtainable.

TABLE II

FAECAL NEMATODIRUS EGG COUNTS IN LAMBS IN SECOND FIELD TRIAL OF BEPHENIUM EMBONATE

250 mg. per kg. bephenium embonate			125 mg. per kg. bephenium embonate			Controls		
Lamb number	Eggs per gramme of faeces		Lamb number	Eggs per gramme of faeces		Lamb number	Eggs per gramme of faeces	
	7.6.57†	12.6.57		7.6.57†	12.6.57		7.6.57	12.6.57
D.4	850	0	V.2	0	0	2	200	0
D.7	600	0	D.13	350	0	V.7	1,250	N.S.*
D.14	250	N.S.	V.18	1,050	0	D.18	950	400
F.14	250	N.S.	D.21	N.S.	0	V.19	1,000	1,150
F.16	2,600	0	V.21	N.S.	0	D.23	50	400
F.17	500	0	D.32	100	0	D.35	800	200
L.18	1,950	0	41	500	200	H.38	600	1,750
F.20	650	0	D.48	N.S.	0	42	900	150
D.22	950	0	F.53	350	0	D.55	2,450	2,250
F.27	200	0	H.55	50	0	D.57	0	0
D.31	800	0	D.59	0	0	F.57	250	450
H.31	550	0	D.62	50	0	V.61	250	0
43	1,150	0	V.66	150	0	F.62	1,050	450
F.50	1,000	50	V.67	150	N.S.	V.62	2,000	650
H.54	550	0	H.68	0	0	D.64	450	650
D.56	650	0	V.68	N.S.	0	H.64	2,000	1,600
H.62	N.S.	0	V.69	N.S.	0	F.71	300	550
H.63	150	50	V.75	650	250	74	450	N.S.
D.67	750	N.S.	V.84	1,400	0	H.77	200	N.S.
H.81	550	0	V.86	150	0	H.79	300	150
D.82	1,100	0	F.87	350	0	82	550	3,400
H.84	350	0	F.89	0	0	V.83	700	750
D.96	550	0	H.89	0	0	F.85	1,400	4,200
D.97	150	0	90	100	0	H.85	500	0
98	300	0	D.90	250	0	H.87	500	1,150
99	550	0	D.92	350	0	D.88	100	0
102	600	250	93	50	0	V.89	950	2,000
D.104	300	0	H.97	750	250	D.94	50	200
D.105	100	0	H.98	2,350	50	D.98	150	450
H.105	250	0	H.99	N.S.	0	D.99	150	850
D.107	1,100	0	D.102	1,400	0	H.101	850	1,200
H.107	500	0	H.104	150	0	D.106	1,150	2,250
D.109	0	N.S.	106	0	0	H.116	1,450	1,750
D.110	N.S.	0	H.108	1,750	1,500	D.117	900	2,400
H.111	650	0	H.113	200	N.S.	H.117	100	300
D.116	2,850	0	D.114	550	350	H.119	0	N.S.
D.119	750	0	H.115	1,300	0	121	600	650
H.120	2,050	0	D.118	N.S.	0	127	50	0
124	900	0	122	1,200	0	128	1,900	1,850
129	150	0	123	400	50	133	350	200
131	1,800	0	125	350	0	134	2,000	3,350
132	300	0	126	1,150	0	140	N.S.	1,450
136	N.S.	0	130	550	0	145	250	1,250
137	750	0	135	300	0	150	50	N.S.
138	0	N.S.	143	1,900	550	155	750	800
139	850	50	149	1,100	150	156	400	1,250
141	2,000	0	153	N.S.	0	158	400	100
142	1,250	0	154	250	0	159	700	2,700
144	600	0	162	50	0	161	1,850	450
146	N.S.	0	163	0	0	171	250	650
148	650	0	166	1,000	150	172	400	1,200
152	1,350	0	170	0	50	173	450	N.S.
164	0	0	174	1,250	0	175	N.S.	1,700
168	0	0	176	1,300	N.S.	177	400	400
169	50	0	178	N.S.	100	179	1,100	650
Averages	740	8		548	70		705	1,028

* N.S. : No sample obtainable. † Immediate pre-treatment.

rates of 500 and 250 mg. per kg. respectively. Unfortunately, as shown by the low faecal egg counts, resistance to the infection was increasing during the trial. This is confirmed by the relatively low numbers of Nematodirus found on *post-mortem* examination of the control animals with the exception of the one lamb (No. 283) which died and was examined by a Veterinary Investigation Officer. There was no evidence, however, in this flock that the 500-mg. per kg. dose was more efficient than that of 250 mg. per kg. The data relating to all lambs with faecal egg counts greater than 300 per gramme before treatment, together with the numbers of Nematodirus in the 3 lambs from each group slaughtered 6 days after treatment, are shown in Table IV. (Page 254.)

In an experiment to determine the effect of

TABLE III

DATA RELATING TO NEMATODIRUS IN LAMBS SLAUGHTERED IN SECOND FIELD TRIAL OF BEPHENIUM EMBONATE

Lamb number	Eggs per gramme of faeces 7.6.57†	Dose (mg. per kg.) administered 7.6.57	Eggs per gramme of faeces		Number of Nematodirus at autopsy, 15.6.57			
					4th larval stage		5th larval stage	Adult
			12.6.57	15.6.57	Early	Late		
43	1,150		0	0	2,850	350	250	350
F.50	1,000	250	50	150	3,350	500	350	3,750
D.107	1,100		0	0	400	50	0	150
V.84	1,400		0	0	0	400	100	0
D.102	1,400	125	0	300	10,059	3,477	3,111	5,847
H.115	1,300		0	0	10,790	3,318	3,052	5,271
V.19	1,000		1,150	N.S.*	13,961	9,137	4,891	50,277
D.106	1,150	Nil	2,250	2,350	6,685	5,909	3,230	14,800
H.116	1,450		1,750	1,800	20,655	3,402	4,158	37,268

* N.S. : No sample obtainable. † Immediate pre-treatment.

TABLE IV

DATA RELATING TO NEMATODIRUS IN SELECTED LAMBS IN THIRD FIELD TRIAL OF BEPHENIUM EMBONATE

Lamb number	Eggs per gramme of faeces, 29.6.57†	Dose (mg. per kg.), 29.6.57	Eggs per gramme of faeces, 3.7.57	Numbers of Nematodirus at autopsy, 5.7.57			
				4th larval stage		5th larval stage	Adult
				Early	Late		
203	300		N.S.*		Not killed		
215	600		0	0	0	0	0
231	400	500	0		Not killed		
234	1,350		0	0	0	0	0
235	550		N.S.		Not killed		
239	650		0	0	0	0	0
300	350		0		Not killed		
312	550		0	0	50	0	0
315	600	250	0	0	50	50	0
316	600		0	0	0	0	0
331	400		0		Not killed		
256	350		150		Not killed		
269	400		100	0	0	50	750
273	500		100	0	0	150	3,850
278	600	Nil	50	100	100	0	3,500
282	300		100		Not killed		
283	600		Died 1.7.57				41,600‡
285	400		0		Not killed		

* N.S. : No sample obtainable. † Immediate pre-treatment. ‡ Examined by a Veterinary Investigation Officer.

bephenium embonate on growth rate, 10 lambs, reared worm-free except for *S. papillosus*, were randomised by weight into a control undosed group, and a group which received 3 doses of 500 mg. per kg. at monthly intervals. The experiment was completed 2 weeks after the third dose was administered, by which time the dosed lambs had gained approximately 10 per cent. more in weight than the controls. This is not statistically significant but illustrates the lack of toxic effect of this dose as judged by weight gain. The results are shown in Table V. Similar data have been obtained in experiments using lambs with mixed infestation of trichostrongyles and, in addition, no clinically-recognisable signs of toxicity have been noted in about 400 lambs treated with single doses varying from 125 to 2,000 mg. per kg.

TABLE V

GROWTH RATE OF "WORM-FREE" LAMBS RECEIVING 500 MG. PER KG. BEPHENIUM EMBONATE AND OF UNTREATED CONTROLS

	Dosed	Controls
Initial average weight (lb.) ...	33·5	33·6
Final average weight (lb.) ...	69·8	66·6
Per cent. average live-weight gain during experiment	108·4	98·2

Discussion

The 3 main species of Nematodirus which are pathogenic to lambs are *N. filicollis*, *N. battus* and *N. spathiger*, the first 2 being important in Britain. Examination of the adult Nematodirus collected at

autopsy of the lambs used in the trials described above revealed only *N. filicollis*. Both *N. battus* and *N. filicollis* were present in lambs used in a field trial conducted by Mr. D. Buntain, Veterinary Investigation Officer, in N.E. England, using bephenium embonate at 500 mg. per kg. and there was no evidence of a differing susceptibility (Buntain, 1957).

The life history of these parasites differs from that of the other trichostrongyles, in that the larvae develop to the infective third stage within the egg. These embryonated eggs may remain dormant on the pasture through the winter to hatch in the following spring (Thomas & Stevens, 1956). This leads to the sudden appearance of infective larvae when lambs are at a susceptible age. When ingested, the larvae undergo a third moult in the small intestine to become fourth-stage larvae, which have been shown in the case of *N. spathiger* (Kates & Turner, 1955) to penetrate and damage the mucosa giving rise to the clinical signs of the disease. A further moult occurs several days later and the fifth stage, the immature adult, emerges to regain the lumen of the small intestine. Outbreaks of clinical Nematodiriasis are believed to arise only when susceptible lambs are suddenly exposed to heavy intakes of infective larvae.

Two methods of control of this disease are therefore indicated. The first is by pasture-grazing management to prevent susceptible lambs from ingesting large numbers of infective larvae. The second is by the correct use of an effective anthelmintic.

Methods of grazing control have recently been discussed (Spedding, 1958). The ideal is to avoid grazing susceptible animals on pasture which carried lambs during the previous year. Grazing control of such stringency is difficult, even impossible, on many farms particularly during the danger period. The second method is to avoid pasture which carried lambs 1 to 4 months old during the previous year and is thus likely to be most heavily infested. Older animals can, however, have high faecal egg counts and on occasion may be the source of heavy pasture contamination (Stamp *et al.*, 1955 ; Kingsbury, 1956). As shown in Table I, high faecal egg counts were recorded in older lambs in September and October, 1956. Of 90 lambs examined in August, 1957, 10 per cent. had faecal counts greater than 1,000 e.p.g. and a further 16 per cent. counts of between 500 and 1,000. Under such circumstances, and particularly when susceptible lambs have to be grazed on pasture heavily infested during the previous season of clinical Nematodiriasis, an efficient anthelmintic is essential.

Some activity against Nematodirus has been claimed for several drugs, but their use in the field has so far been disappointing. Thus, Turner & Colglazier (1954) found that, in lambs suffering from naturally-acquired mixed infestations of *N. spathiger* and *Haemonchus contortus*, free access to a 1 in 9 phenothiazine-salt mixture reduced the numbers of both parasites. There are no reports on the effect of free access to phenothiazine-salt mixtures on *N. battus* and *N. filicollis*, though phenothiazine has been found to have about a 50 per cent. efficiency in lambs experimentally infected with these species (Report on the Animal Health Services of Great Britain, 1955). However, the usual therapeutic doses of phenothiazine have not been effective in controlled trials (Imperial Agricultural Bureaux, 1943) or in general use. Similarly, hexachloroethane (Kingsbury, 1953) and piperazine citrate (Rowlands, 1955) appeared to have some efficiency in the original trials, but this has not been substantiated. A chlorinated aliphatic hydrocarbon mixture has more recently been claimed to give a good clinical response (Bywater, 1957). This preparation was administered to 8 lambs in the flock used for the second field trial of bephenium embonate, but the faecal Nematodirus egg counts of these lambs rose after treatment as did those of control untreated lambs.

Bephenium embonate, because of its effect, at the 250-mg. per kg. dose against the larval and adult stages of Nematodirus, should prove of great benefit in the prophylaxis and treatment of this infestation. The epidemiology of the disease suggests that, for prophylaxis, in the normal season of heavy infestation, the drug should be administered at approximately 3-weekly intervals, at the beginning and end of May and again in mid-June, and that the benefit obtained should be cumulative in the following year.

Acknowledgments.—We would like to express our thanks for the help received from the following collaborators :—

(1) Numerous colleagues in the Wellcome Foundation who were associated with this work, in particular Dr. F. C. Copp who prepared bephenium embonate ; Dr. O. D. Standen who assayed the material biologically in laboratory animals ; Mr. D. Stephenson who provided materials ; Mr. S. L. Hignett for his encouragement throughout, and Miss W. A. Morley and Miss G. Wrapson for technical assistance.

(2) The veterinary surgeons in the Veterinary Investigation Service, Mr. D. Buntain and Mr. J. D. Paterson, and those in private practice who assisted in these and other trials.

(3) The farmers who made lambs available and provided staff to help with this work.

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Refresher Article No. 6

Coccidiosis in Domestic Mammals

BY

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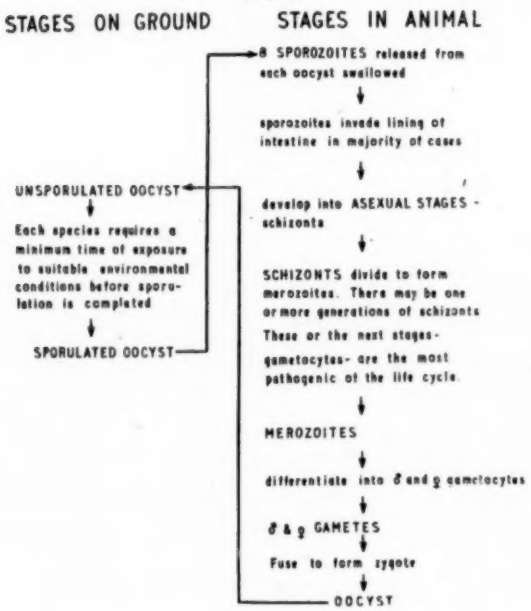
ALL domesticated mammals, with the exception of the horse, carry species of coccidia which can, under appropriate circumstances, produce disease. They also carry a number of coccidial species which appear to do little harm, even when they are present in considerable numbers. With the single exception of *Eimeria stiedae* which passes its trophic phases in the liver of the rabbit, all the mammalian *Eimeriae* are inhabitants of the intestinal tract. The distribution of these intestinal species varies and different species tend to show a preference for different parts of the intestine, although there is frequently an overlapping of the range of one species with another. The coccidia exhibit a host-restriction and it is only rarely that an infection can spread from one species of animal to another.

The life histories of the various species of coccidia differ in detail but broadly they follow a common plan. A coccidial infestation is initiated after the host animal ingests an infective or sporulated oocyst of a species of coccidium that is capable of developing in it. A condensed representation of the life history is shown in Fig. 1. It will be seen from the diagram that the oocyst is the culmination of the coccidial life history and as such represents the end of a line of parasite reproduction and a lessening of the extent of parasite activity within the host. Provided an infected host animal is prevented from ingesting more oocysts a coccidial infestation will eventually end with the discharge of oocysts in the faeces. The coccidial life history is therefore a self-limiting one.

FIG. 1.


STAGES ON GROUND
STAGES IN ANIMAL
8 SPOROZOITES released from each oocyst swallowed
sporozoites invade lining of intestine in majority of cases
UNSPORULATED OOCYST
develop into ASEXUAL STAGES - schizonts
Each species requires a minimum time of exposure to suitable environmental conditions before sporulation is completed
SCHIZONTS divide to form merozoites. There may be one or more generations of schizonts
These or the next stages - gametocytes - are the most pathogenic of the life cycle.
SPORULATED OOCYST
MEROZOITES
differentiate into ♂ and ♀ gametocytes
♂ & ♀ GAMETES
Fuse to form zygote
OOCYST

Sporulation time at given temperatures is more or less constant for each species of coccidium and may differ between species; consequently, sporulation time can often serve as one of the several characters used in determining their identity. The time elapsing between the ingestion of an oocyst and the production of a new generation of oocysts (the prepatent period) is again conditioned by the species of coccidium and thus provides more evidence of its identity. Other characters used for identifying species are the morphology of the unsporulated and sporulated oocyst, the site in the intestine at which the infestation is concentrated, as well as the kind of disease produced by pathogenic species. All species of coccidia, if present in sufficiently large numbers, induce demonstrable immune responses in their hosts; no cross-immunity is developed between the different species of coccidia of one species of host animal or, of course, between those of different host animals. This quality of specific immunity can be used as a further guide to the identity of the coccidium.

It will be realised that the presence of oocysts in the faeces of a host represents a certain amount of damage done to the intestinal epithelium of the host and, in general, the higher the number of oocysts the greater the damage done. On the other hand, some species of coccidia are more important than others from the standpoint of disease, and the presence of large numbers of oocysts of certain species in the faeces may mean little from the standpoint of disease; in passing, it must be noted that some damage to the intestinal epithelium accrues from any coccidial infestation, irrespective of species, but in some cases the damage done is greater than in others. The possibility that the so-called non-pathogenic coccidia do cause some deterioration in the host cannot be dismissed and such deterioration may reveal itself in animals that are substandard in the matter, for example, of growth rate. Several examples of such a condition are to be found in fowls parasitised by coccidia that are only rarely lethal and which are usually regarded as harmless.

Although a similar condition exists in mammals, investigation of the effects of this sort of parasitism and the species of coccidia concerned in it is required before any generalisations can be made.

It will be appreciated that a definite diagnosis of clinical coccidiosis can only be made in the light of our knowledge of the kinds of coccidia which might produce the observed conditions. In mammalian coccidiosis it is often possible to determine, with fair accuracy, the identity of the coccidia by reference to the oocyst characters. Before going further it can

be assumed that, in general, young animals carry coccidia of one or more species. Where mammals are the natural hosts for a number of different species of coccidia it is essential for the diagnostician to recognise the predominant species in a mixed infestation. Since some animals may carry as many as a dozen species of coccidia, some being possible causal agents of really serious and often fatal disease, whereas others are non-killing and generally less pathogenic in their effects, it is simpler for the non-specialist to familiarise himself with the commoner and more serious coccidial diseases and the species of coccidia with which they are associated.

These coccidial diseases may arise in susceptible young animals in contact with carrier young or adult animals with low-grade infestations. There is a rapid multiplication of parasites in the young animals, and other susceptible young animals in contact with them may develop subclinical infestations in which the symptoms are less severe than those occurring in the clinical cases which are well-defined and simpler to diagnose. These subclinical cases may exhibit poor growth and diarrhoea without blood; such infestations produce large numbers of oocysts, and young susceptible animals brought into contact with such cases are likely to become seriously diseased and die. The numbers of oocysts will, of course, increase greatly in over-crowded pens which are not cleaned out regularly. Bedding, food, and water contaminated with the faecal matter of parasitised animals are common sources of infection. Animals can also become infected by licking the soiled coats of infected animals; this source of infection may be commoner than appears, and it should be pointed out that the temperature and moisture of such situations are conducive to the rapid sporulation and survival of oocysts. Naturally, temperature or any other factor (*e.g.* moisture) of the oocyst environment which may condition their rate of sporulation will also influence the pattern of epidemics.

Coccidiosis in Cattle

Of the many species of coccidia isolated from British cattle probably only three are possible causes of serious disease. To avoid confusion no mention will be made of the other, less pathogenic species.

Diarrhoea is the most typical symptom associated with coccidiosis of cattle; others are weakness, emaciation, inappetence, and anaemia. The diarrhoea is not necessarily bloody. In the less advanced disease the discharges are brownish or greyish in colour, and watery. In the more advanced cases of heavy infestations, strings of mucus and blood may be discharged at frequent intervals and tenesmus is pronounced. Examinations of rectal samples will usually reveal numerous oocysts although an animal may die as a result of a single heavy infection before oocysts are formed, in which case merozoites may be found.

Provided the oocysts of the severely pathogenic coccidia can be recognised, a heavy yield of these oocysts on salt flotation or smear may be taken as suggestive of disease, when there is also evidence of unthriftiness and/or diarrhoea. Should a count be made by one of the conventional methods anything above 5,000 oocysts per gramme of faeces is suspect; allowance must, of course, be made for the fluid content of diarrhoeic faeces, as well as the species of coccidium concerned.

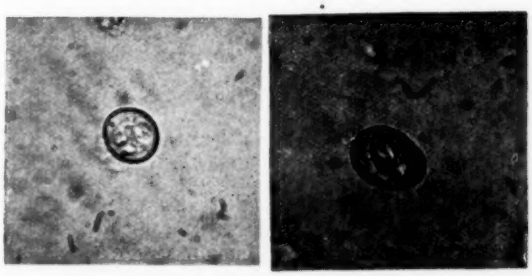
Examination *post-mortem* of the intestinal tract shows a catarrhal enteritis in the small and large intestine. In severe cases, destruction of the epithelium with thickening of the mucous membranes are evident. Small, greyish patches, with or without diffuse haemorrhages or petechiae, may be seen in the small intestine and caecum, or whitish ridges in the rectum. The presence of scattered, small white spots in the villi may be detected in the small intestine. Microscopical searches for parasite stages should be made by taking fresh smear preparations of the intestinal contents and mucosa. The intestinal contents will usually reveal merozoites and oocysts, and the mucosa the intracellular stages of the life-cycle, the schizonts, merozoites, and gametocytes.

The pathogenic coccidia of bovines include *Eimeria ellipsoidalis*, *E. bovis* (syn. *E. smithi*), and *E. zurnii*. It is convenient to compare the sizes of these oocysts with that of the bovine red blood corpuscle. The oocyst of *E. ellipsoidalis* is small and ellipsoidal in shape and is approximately three times as large as the diameter of the red blood corpuscle. The oocyst of *E. bovis* is large and egg-shaped, although considerable variations in shape occur (Fig. 2B); in colour the oocyst appears greenish to light brown under low powers of the microscope; it measures approximately 5 to 6 times the diameter of the red blood corpuscle. The oocyst of *E. zurnii* is spherical or slightly subspherical in shape and in diameter measures just over three times that of the red blood corpuscle. The sporulation times of the oocysts of these species vary from 48 to 72 hours in each case. It is interesting to recall that the sporulation of the oocysts of some of the less common coccidia of cattle may take as long as 6 or 7 days.

Eimeria ellipsoidalis can cause diarrhoea with little or no blood in calves of 1 to 3 months of age. The intracellular part of the life cycle is completed in the mucosal epithelial cells of the small intestine. In passing, it might be noted that a species not so far mentioned, *E. cylindrica*, is regarded by some as slightly pathogenic and is said to produce blood-stained faeces.

FIG. 2.

A B



E. zurnii oocyst. x 400. *E. bovis* sporulated oocyst. x 400.

E. bovis produces a severe form of coccidiosis which can usually be anticipated when large numbers of oocysts are found in the faeces of calves of up to 3 months of age. This species is most commonly found in older cattle of over 5 months of age, but disease is less common in this age group. Under experimental conditions calves usually begin to show symptoms 18 days after the administration of oocysts. At this time the faeces become diarrhoeic and blood-streaked. The severity of the condition increases during subsequent days and frequent discharges of bloody fluid and blood clots are mixed with the faeces. The animals soon cease to feed, and later frequent tenesmus, with only an occasional discharge of blood and fragments of tissue, occurs. Dehydration and extreme weakness develop and the calf may die within one month of administering the infective dose of oocysts. At examination *post mortem*, bloody fluid is found in the posterior part of the ileum, the caecum, and colon. There is noticeable thickening of the walls of the caecum and colon, the contents of which contain blood; pieces of sloughed mucosal epithelium are often present. Smears of the contents will reveal oocysts and other stages. The white spots already mentioned as occurring in the villi of the posterior parts of the small intestine are large schizonts situated in the central lacteals and which have often been mistaken for *Globidium* and *Gastrocystis*. There is a single asexual generation in the life cycle of *E. bovis* and, as may be the case with other species with a single asexual generation, the sexual stage is the pathogenic one. In species with two asexual generations it is generally the second one that is markedly pathogenic. The oocysts of *E. bovis* are found chiefly in the glands of the caecum and large intestine.

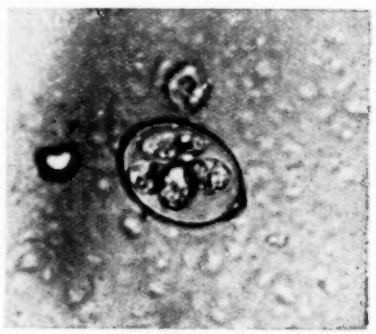
E. zurnii lives in the small and large intestine and is the cause of "bloody diarrhoea" in cattle, and is regarded as the predominantly pathogenic coccidium of cattle. Microscopic examination of the discharges usually reveals enormous numbers of the spherical, greenish-violet tinted oocysts (Fig. 2A). The symptoms of the disease, apart from the bloody diarrhoea, are anaemia, extreme weakness and emaciation, and straining accompanies defaecation. As in all forms of intestinal coccidiosis great damage is done to the intestinal walls by the rapidly multiplying and growing stages of the coccidial life cycle. Examination *post mortem* will show considerable sloughing of the intestinal epithelium, petechial or diffuse haemorrhages, and catarrhal enteritis. The most marked pathological changes are found in the large intestine and it is in the glandular epithelium of this region that large numbers of asexual and sexual stages will occur.

In condensing this consideration of the several forms of coccidiosis occurring in cattle certain generalisations can be made. Acute disease is usually associated with the crowding together of young animals. Coccidiosis, whether in mammals or birds, has only reached its considerable economic importance since the mass production of animals has necessitated high concentration of them within the smallest possible space. Calves confined in pens became more heavily infected with coccidia than calves on pasture. Outbreaks of disease in young or susceptible older animals kept in crowded quarters result from at least two factors; one is that disease occurs in animals which have less resistance to parasitism than other previously infected ones, and the second is that such animals are exposed to a large number of parasites. These factors are also concerned in the development of coccidiosis in the other species of domestic animals.

Coccidiosis in Sheep

Coccidiosis occurs in British sheep but there has been little published work describing the species of coccidia responsible for the disease. *Eimeria arloingi* has been incriminated in several outbreaks of coccidiosis in Britain and it is likely that this species may be the chief cause of severe ovine coccidiosis in these islands. This view of the economic importance of *E. arloingi* is a widely held one among workers in coccidiosis outside this country, and mortality and deaths have been reported as resulting from heavy infestations. Other species which occur in Britain are *E. intricata* and *E. parva*. Other species have also been observed but no precise identifications have been made. For our present purpose it will probably be sufficient to record what is known about the disease caused by *E. arloingi*. So far as is known, *E. intricata* has not been implicated in any case of ovine coccidiosis in Britain; the status of *E. parva* is somewhat doubtful. Cases have occurred in Britain in which young animals under one month of age have suffered from scouring and weakness, and large numbers of oocysts resembling those of *E. parva* have been demonstrated in their faeces. Deaths resulting from heavy infestations with this species have been reported in other countries. The oocysts of *E. intricata* are unmistakable. They are the largest oocysts occurring in sheep and measure from 40 to 60 μ in length. The oocyst wall which is thick, opaque, and striated, is brown in colour and a pronounced polar cap is present. The oocysts of *E. parva* are, on the contrary, subspherical, small, and measure up to 22 μ in length; no polar cap can be detected.

FIG. 3.



E. arloingi. Sporulated oocyst. x 600.

The oocyst of *E. arloingi* (Fig. 3) is ovoid and it may measure anything from 17 to 42 μ in length and there is a prominent polar cap. This species may cause disease in lambs of from 2 to 6 months of age. Anaemia, loss of weight, and yellowish, blood-stained diarrhoea are suggestive of the condition, and there may be deaths. Examination of dead animals will reveal enteritis, particularly in the small intestine, and whitish, discrete spots or yellowish diffuse areas will be evident. The spots can be detected through the serosa. If crush preparations of these spots are made and examined under the microscope, large numbers of gametocytes and oocysts will be seen. The asexual stages which appear to be the most pathogenic ones are found in the villi.

An epidemic may be initiated by the introduction of a carrier animal into an environment which permits the accumulation of viable oocysts such as, for example, crowded, damp pastures. American workers consider that feed contaminated by fresh faeces is a common source of infestation. Chopped feed, kept in open troughs which are not regularly cleaned, may provide such a source.

The view has been expressed that much of the morbidity occurring during ovine coccidiosis may result from toxicity and that the toxin may be a product of the coccidial infestation.

Coccidiosis in Goats

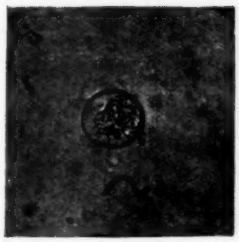
Although it is generally assumed that goats are susceptible to infestations with the same species of coccidia as sheep, there is, in fact, very little experimental proof of this assumption. It cannot be denied that goats do suffer from coccidiosis, and cases have occurred in this country. Superficial examination may certainly reveal oocysts similar to those of sheep coccidia, but whether or not they are really identical species is purely a matter of academic interest, since satisfactory results are obtained by treating coccidiosis of goats in the same manner as the disease in sheep.

Coccidiosis in Pigs

The incidence of coccidiosis in pigs in Britain is difficult to assess. However, the examination of pig faeces has shown what were believed to be the oocysts of *E. debliecki, E. scabra,* and *E. perminuta. Isospora suis* (the oocyst of *Isopora,* in contrast to that of *Eimeria,* has only two sporocysts, which contain four sporozoites each) has not been seen by the writer. In view of the small numbers of samples examined, it is quite possible that these and other species are more widely distributed than is at present appreciated.

E. debliecki and *E. scabra* produce several asexual generations. The oocysts of *E. debliecki* are of variable dimensions ranging from 12 to 30 μ in length and they require a long time, 7 to 9 days, for the completion of sporulation. They are smooth walled and ovoidal and have no micropyle. The oocysts of *E. scabra* are ellipsoidal, rough walled, and sporulation may take as long as 12 days. They measure anything from 22 to 36 μ in length. Diseased pigs rarely, if ever, pass copious blood in their faeces, but severe diarrhoea occurs which is often followed by emaciation and death. Sloughing of the epithelium of the large intestine has been described in heavy infestations. Morbidity frequently persists in recovered animals.

FIG. 4.



I. bigemina. Unsporulated oocyst. x 400.

I. bigemina. Sporulated oocyst. x 400.

Coccidiosis in Dogs and Cats

Coccidiosis in the dog and cat is best considered as one disease, since they have much in common. This does not mean that the diseases themselves are necessarily caused by the same organism. The genus *Isospora* is the important one although both dog and cat may harbour different species of the genus *Eimeria.* The distinction between these genera has already been alluded to in the section on pigs.

Canine coccidiosis occurs in Britain, and sometimes on a substantial scale where numbers of dogs are confined together. The position in cats is less clear.

Isospora bigemina is the species typically concerned in canine coccidiosis. The oocysts are spherical or ovoidal in shape and have no micropyle (Fig. 4). They appear in two forms, large (18 to 20 μ in length) and small (10 to 14 μ in length), although these two extremes are linked together by oocysts of varying size. These two forms do not seem to occur together in the same infection. A character that is exceptional among the coccidia of other warm-blooded animals (although it is met with in cold-blooded vertebrates) is the passing of sporulated oocysts or isolated spores by subclinically infected dogs. In acute disease only unsporulated oocysts are discharged; these will sporulate in 4 days.

Asexual multiplication takes place in the epithelial tissues of the small intestine where a haemorrhagic enteritis, and, in severe infestations, ulceration occur. The severest lesions are to be found in the ileum. Haemorrhages may be diffuse or petechial according to the severity of the disease. Large-scale desquamation of the epithelium may occur together with mucosal thickening.

Diarrhoea, anaemia, emaciation, and weakening are the usual symptoms of disease. The faeces may become mucoid and haemorrhagic. The animal becomes depressed and evinces pain on palpation.

Other species which apparently produce much the same condition as *I. bigemina* are *I. felis* and *I. rivolta.* The oocysts of *I. felis,* which are the largest ones found in dogs and cats, measure as much as 39 to 48 μ in length. These species appear to enjoy

a less rigid host-restriction than is usual among coccidia.

I. bigemina (or possibly variants of this species) occurs in the dog and cat as well as certain other carnivores like foxes and polecats.

I. felis. Variants are reported from dog, cat, lion, and serval.

I. rivolta has been recovered from the dog, cat, and dingo.

A species, *I. hominis,* has been regarded as a parasite of man; it is possible that it was actually *I. bigemina* var. *canis* which had been accidentally swallowed by a none too particular dog lover! In fact some authorities consider that another species, *I belli,* is the only true coccidium of man.

Coccidiosis in Rabbits

The species of coccidia described from the rabbit are now so numerous that space will only permit a consideration of the more pathogenic species. These are mostly restricted to the rabbit and recent Hungarian work indicates that the hare probably plays no great part in carrying intestinal coccidiosis to domestic rabbits. On the other hand, the liver-inhabiting *E. stiedae* is less restricted and in one or two instances appears to have been transmitted from rabbit to hare although its natural occurrence in wild rabbits and hares appears unusual.

Routine faecal examinations for oocysts are not always helpful since different species of coccidia may produce oocysts with only slight morphological differences, *e.g.* those of *E. stiedae,* and *E. irresidua.*

FIG. 5.

E. stiedae. Unsporulated oocyst. x 400.

E. stiedae. Sporulated oocyst. x 400.

At examination *post mortem* the presence of *E. stiedae* can be confirmed by taking a sample of the contents of the gall bladder. Any oocysts present can be assumed to be those of *E. stiedae;* in practice, of course, the liver lesion is diagnostic. There are a number of species of coccidia in British domestic rabbits but we have little information about the incidence and pathogenicity of most of them. There is, however, little doubt that *E. stiedae* is responsible for serious disease in rabbit colonies. Other outbreaks in Britain have been caused by the intestinal species, *E. magna,* to a lesser degree by *E. irresidua* and *E. perforans.* Like other forms of mammalian coccidiosis, the disease is typically one of young animals, and often attacks them just after weaning.

E. stiedae parasitises the epithelial cells of the bile ducts where the asexual and sexual cycles are passed. White nodules which are filled with oocysts appear on the surface of the liver. The oocyst wall may be pink, orange, or brown in colour, and is ovoidal or ellipsoidal in shape. A micropyle is present at the narrower pole. They are large oocysts of 28 to 42 μ in length and 16 to 25 μ in breadth (Fig. 5). In severe infestations the parasites cause considerable enlargement of the liver which gives the pot-bellied appearance so commonly seen in rabbits with this disease.

E. magna is a parasite of the small intestine. The oocyst wall is brownish in colour and is broadly ovoidal in shape. The micropyle is broad and distinct and its margins are quite characteristic, since they show, sometimes prominently, knob-like irregularities. The oocyst is large and measures from 28 to 42 μ in length and from 20 to 26 μ in breadth. Biologically speaking, this is an interesting species which can infect and produce clinical coccidiosis in cottontails, and in which the prepatent period averages 4 days, and the patent period 14 days. The prepatent period rose from 6 to 7 days and the patent period from 12 to 21 days when identical dosage and culture were administered to domestic rabbits. The dimensions of the oocysts derived from the two species of hosts also differed. The condition of the *E. magna*-infected rabbits in Britain is similar to that described in cottontails, *i.e.* rough coat, inappetence, softened pellets, followed by severe diarrhoea and death.

E. irresidua and *E. perforans.* The symptoms associated with these species are similar to those produced in infestations with *E. magna.* The oocysts of *E. irresidua* are dark yellow in colour and ovoid in shape. There is a distinct concave micropyle and a thickening of the oocyst wall in this region. They measure from 31 to 43 μ in length, and 22 to 27 μ in breadth. The oocysts of *E. perforans* are oval and equally rounded at both ends. There is no micropyle and the wall may be irregular in contour. The oocyst ranges from 15 to 30 μ in length and from 11 to 20 μ in breadth. Adult carriers are sources of infestation. Young rabbits frequently soil their coats by lying on faecally-contaminated bedding. In the process of grooming themselves, faecal matter, containing oocysts, is ingested and an infestation is initiated. The swallowing of night faeces obtained directly from the anus, as practised by rabbits, is of little importance as far as coccidiosis is concerned since any oocysts swallowed with the faeces will be unsporulated and therefore non-infective. In heavy infestations with intestinal species, it is conceivable that merozoites in the night faeces may be re-introduced into the animal by re-ingestion, but there is no proof of this happening.

The Coccidia of Laboratory Animals: Rats, Mice, Guinea-pigs, and Ferrets

Host-specific coccidia occur in rats and mice. *E. miyairii* lives in the small intestine, caecum, and colon of the rat, and severe infestation may cause death.

E. falciformis is a parasite of the small intestine, and *Cryptosporidium muris* a parasite of the stomach

of mice. The guinea-pig carries *Eimeria caviae*. The importance as disease entities of the coccidia of the mouse and guinea-pig is an open question. The ferret is known to harbour three species: *Isospora laidlawi*, and more frequently *E. ictidea* and *E. furonis*, all of which occur in the intestine. *E. ictidea* invades only the free ends of the villi and sometimes only their summits, thus leaving the basal portions uninfected. This species tends to constrict off the infected parts of the villi from the non-infected parts; this is an interesting observation which may be unique in the pathology of coccidiosis.

The more important species of coccidia have now been considered in their relation to the production of disease in domestic animals. It is fully appreciated that the practising veterinary surgeon is more concerned with the recognition of coccidial diseases and their treatment than with details of the coccidial life histories and species. The recognition of disease is obviously important in making a diagnosis of disease in an obviously sick animal, but where animals are kept together the group and not the individual must be considered the potential patient. Most of the contact animals may appear normal but may develop disease later; it is necessary, for satisfactory control, to be familiar not only with the symptoms of disease and their treatment, but also with the broader epidemiological aspects of the disease and the features to be expected and revealed at examination *post mortem*. Having now considered some of the salient features of the mammalian diseases of coccidial origin something must now be said about the methods by which they may be prevented and treated.

Control of Coccidial Diseases in Domestic Animals

(a) *Prevention*

Several important facts emerge from what has been said about the forms of coccidiosis occurring in domestic animals and the life-cycles of the protozoa causing them. First, the adult carrier animal which may carry substantial burdens of coccidia without harm to itself may prove to be a definite hazard to young stock. The young animals may become infected by eating contaminated bedding or food, by licking their soiled coats or those of the adults, or faecal splashes on floors or walls. So far as is practicable, young stock should be separated from adult stock. Crowding of young stock will tend to build up the oocyst population in their environment when an infestation is present in one or more animals. Under normally good farming conditions, patent disease develops from reinfestation rather than from a single infestation. Every effort must be made to reduce re-infestation to small proportions which will encourage resistance to develop in young animals. Infestations are initiated when animals pick up sporulated oocysts. As known, the progress of sporulation requires a minimum time on the ground for its completion and the length of time required is largely conditioned by the species of coccidium and temperature and moisture of the oocysts' environment. The time elapsing between the passing of oocysts in faeces and their development to infectivity is usually sufficiently long to allow for the removal of contaminated bedding from contact with the animals before the oocysts become infective. The oocyst, as the end product of the life-cycle, does, by its discharge in the faeces, reduce the state of parasitism in the animal host which will, provided it is not re-infected, eventually rid itself of all coccidia.

As a result of a single lethal infestation, which is probably rare in nature, a host may die before oocysts are formed, since the lethal stages of the life-cycle are usually the late asexual and early sexual ones. The viability of oocysts depends on the presence of moisture in their environment so that the aim of the farmer should be to preserve as dry a condition as possible in the animal quarters. Much can be done by grooming the animals and by practising good hygiene in their management.

(b) *Treatment of Diseased Animals*

Should preventive precautions fail and animals become diseased, there are various drugs which can be used in the treatment of coccidial diseases. On the whole, coccidiosis of farm animals responds well to sulphonamide therapy.

Eimeria bovis and *E. zurnii* infestations in cattle can often be controlled even if medication is delayed until the symptoms of coccidiosis are pronounced. Naturally the speed of recovery is retarded if treatment is delayed too long. Sulphadimidine* has, in the writer's experience, proved eminently successful in dealing with both these infections. The recommended schedule of treatment consists of an initial dose of 0.1 gramme per kilo bodyweight and maintenance doses of 0.05 gramme per kilo given daily for from 3 to 5 days. A similar dosage is also applicable to sheep, goats, and probably pigs. Other drugs that have been reported as being valuable are 4-4'-diaminodiphenyl sulphone (for *E. zurnii* infection) at the rate of 4 grammes per 100 lb. bodyweight every 24 hours for six days, phthalylsulphathiazole administered as 5 to 7.5 grammes per 100 lb. bodyweight daily for 3 to 4 days, mepacrine hydrochloride at 1 gramme per 200 lb. bodyweight as a drench used in two parts administered daily for 2 to 4 days, sulphaguanidine is especially valuable in the preventive treatment of contact calves when it is used in daily doses of 60 mg. per kilo bodyweight for 3 or 4 days. Sulphaquinoxaline used at the same daily dosage or sulphaguanidine for from 3 to 5 days will also control bovine coccidiosis. In all these cases sick animals should be treated individually to ensure that each receives its full therapeutic dose.

Nitrofurazone incorporated in the food at 0.04 per cent. has been reported to be of value in the treatment of coccidiosis in pigs. The drug has also been stated to be effective against coccidiosis in sheep and goats when administered at a dosage of 10 mg. per kilo bodyweight per day for seven days.

Dogs. Sulphadimidine has proved of value in controlling the disease in dogs. An initial loading dose of 0.2 g. followed by maintenance doses of 0.05

* Sulphamezathine.

(*Continued at foot of col.* 1, *page* 262)

A Case of "Feline Enteritis" Treated with Cortisone and ACTH

BY

CLAUDE NOBLE and MYRE SIM

SUMMARY.—(1) *The problem of differential diagnosis of "feline enteritis" is discussed.*

(2) *A case regarded as "feline enteritis" in a Siamese cat, together with its treatment with cortisone and ACTH, is reported.*

(3) *The dramatic recovery of the animal and the rationale of the treatment is discussed.*

(4) *The possibility of acute virus infection suppressing adreno-cortical function is considered.*

Introduction

THE diagnosis of feline enteritis is traditionally subdivided into three clinical groups: (a) the hyperacute, (b) the acute, and (c) the sub-acute (Curasson, 1946). Siamese and Persian cats are most vulnerable and rarely survive the acute form, while the hyperacute form is regarded as invariably fatal in a matter of hours. It is reputed to be highly infectious and due to a filterable virus; treatment of groups (a) and (b) is not very effective, though claims have been made for antibiotics and pyrogens, especially in group (b), and a vaccine has also been prepared. There is some doubt as to the full identity of the disease and Gledhill (1952) considered that the disease in the United Kingdom is the same as feline infective leucopaenia in the U.S.A. This label merely substitutes a laboratory finding for information obtained through clinical examination. The leucopaenia is not an aetiological factor, neither is it a specific manifestation of the disease; it is itself caused by a variety of agencies and apart from the modern trend to prefer laboratory tests to clinical evaluation, has little real advantage over the term "enteritis."

Other acute alimentary disorders could be mistaken for "feline enteritis" but as the illness observed was in a breed of cat notoriously susceptible to it, and since there were a number of other cats in the neighbourhood suffering from a similar disease, indicating an epidemic, it was felt justifiable to regard it as "feline enteritis." It may be that in the hyperacute form, acute alimentary allergy is a complication in a way analogous to gastric influenza in man, and that the disease observed was a sporadic form of this allergy, but without all the resources of a virus laboratory, this could only be a matter for speculation. To the ordinary veterinary practitioner diagnosis will depend on what is the likeliest under the circumstances, and in this case we felt that "feline enteritis" was an appropriate label. This, however, does not signify satisfaction with the present diagnostic state and further differentiation of these diseases is necessary.

Case Report

The following is a report of a cat that contracted the disease. The patient, a Siamese pedigree male (neutered), aged 9 months, and weighing 3 kilos, had been inoculated at 4 months by a reputable feline enteritis vaccine. He was perfectly well on the evening of March 14th, 1957, and ate his supper as usual at 11 p.m. By 7 a.m. the following day he had vomited repeatedly and was incontinent of urine and faeces in his basket. He was lying in the mess, and the faeces were diarrhoeic in nature. He was grossly dehydrated, being too weak to stand, and just lay where he was, obviously distressed at being unable to move. Feline enteritis of the hyperacute group was diagnosed and as a Siamese cat in the same household had died a year before after liberal doses of antibiotics and saline infusions, it was felt that other than replacing the fluid loss there was little that could be done.

While the diagnosis was probably that of an acute virus infection, there were features very reminiscent of an acute adrenal failure, showing rapid physical deterioration, with anorexia, vomiting, diarrhoea, dehydration, and muscular weakness (Cecil & Loeb, 1955; Chester Jones, 1957). One of us (M.S.) suggested that cortisone, if given quickly and in adequate dosage, might help, and with 20 c.c. of glucose saline*

* Intradex.

Coccidiosis in Domestic Mammals.—*Concluded.*

g. given morning and night for 3 days is recommended. The same schedule of treatment can be used in cats.

Rabbits. Therapeutic control of naturally occurring epidemics of *E. stiedae* in weaned rabbits is obtained by giving 0.1 per cent. sulphaquinoxaline in the drinking water or feed continuously for 2 weeks. Sulphadimidine in concentrations of 0.5 to 1.0 per cent. can be used for prophylaxis and treatment. Sulphamerazine (0.5 gramme given orally twice daily for 2 days) and sulphadimidine have been used successfully in controlling intestinal coccidiosis. Sulphaquinoxaline given in the feed or water at a concentration of 0.1 per cent. for 14 days has proved effective. Workers in the Belgian Congo have found nitrofurazone of value in controlling coccidiosis caused by *E. irresidua* and *E. perforans*. Others have found it of little value in treating hepatic coccidiosis.

Rats, mice, guinea-pigs, and ferrets. Little or no work has been reported on the chemotherapy of coccidiosis in these species, but in view of the wide and successful use of sulphonamides in controlling coccidial infections of various sorts it is likely that these compounds will also control infestations in these species.

Chemotherapy in no way replaces good hygiene, and should only be used as a second line of defence.

Finally, it must be emphasised that the demonstration of oocysts in the faeces is not a firm foundation for believing that an animal is suffering from coccidiosis; neither does the absence of oocysts in the faeces necessarily eliminate coccidiosis; cautious appraisal of the clinical findings and history must take first place in making any diagnosis.

intravenously and 40 c.c. of subcutaneous saline, an intramuscular injection of 25 mg. of cortisone was given at 10 a.m. By 2 p.m. the cat could stand and take a little milk and egg and keep it down, and remained fairly comfortable. Towards evening, however, he began to relapse and was apathetic, restless, and could not stand, and another 25 mg. of cortisone was given at 10 p.m. At 3 a.m. the following day he uttered a familiar cry, made his way upstairs, and when presented with a saucer of milk, lapped it up, and rapidly gained in strength.

The cortisone was repeated that day, 25 mg. being administered at 10 a.m. and at 10 p.m., and for the next 3 days it was reduced to 12.5 mg. and given in conjunction with 10 units of ACTH in a single injection at 10 a.m. The cat rapidly regained his lost weight and energy, and great difficulty was experienced in containing him in the house during the 3 days he was being weaned from the cortisone. He has since remained in perfect health, apart from the usual slight discomfort during the hot summer months.

Discussion

From the strictly scientific aspect, all one can say is that comparatively large doses of cortisone can be given to a desperately sick cat without killing it, yet the sequence of events does suggest that the cortisone played some part in the dramatic recovery. In man, adrenal apoplexy is usually associated with meningococcal septicaemia and haemorrhage into the suprarenals (Waterhouse-Friderischen Syndrome) (Greene, 1948). In this acute virus infection one is led to speculate that there may be a sudden suppression of adrenal-cortical function and that it is this aspect of the disease which causes rapid deterioration and death. Such a hypothesis should be capable of proof or disproof, though the virulent nature of the disease would no doubt be a powerful deterrent to anyone introducing an infected animal into a laboratory with other animals.

References

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CHESTER JONES, I. (1957). "The Adrenal Cortex." Cambridge University Press.
CURASSON, G. (1946). "Maladies infectieuses des animaux domestiques." Vigot Frères, Paris.
GLEDHILL, A. W. (1952). *Vet. Rec.* **64.** 723-7.
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Sewage-grown Algae as a Feed-stuff for Chicks. GRAU, C. R., and KLEIN, N. W. (1957). *Poult. Sci.* **36.** 1,046-52.

The authors describe some preliminary experiments in which unicellular algae such as Chlorella and Scenedesmus were grown in sewage, separated by alum flocculation or by centrifugation, and fed for 8 days to 10-day-old chicks. The algae showed a crude protein content of from 31 to 43 per cent. The chicks tolerated as much as 20 per cent. of algae meal prepared by centrifugation, but only 10 per cent. when residual alum was present. Neutralisation and acid extraction were unsuccessful in removing the harmful effects of the aluminium.

E. A. G.

Current Literature

ABSTRACTS

Brucella suis Infection of a Child in Eire. WILLIAMS, T. P., ENTWISTLE, D. M., MASTERS, P. L., & WOODS, A. C. (1957). *Lancet*, December 14th. 1,203–4.

This is the first report of human infection by Brucella suis (American type) in the British Isles. Human infection with *Br. abortus* is sporadic in spite of the widespread infection of cattle. *Br. melitensis* has been isolated from cattle and from their milk in various parts of Great Britain since 1940 but Cruickshank stated in 1954 that it had not been isolated from man in Britain.

Br. suis has been found mainly in the Middle-West of North America, Brazil and Argentina where it infects both pigs and cattle. The isolation of *Br. suis* reported as nearest to Britain is that of *Br. suis* infection of pigs in Switzerland and of milk of a cow.

The patient in this report was a boy aged 4 years who, though a Londoner, spent a holiday with his family on a farm in Eire. The boy became ill after staying on the farm for between 2 and 3 weeks. There were cows on the farm and all the family drank unheated milk from them. Pigs on the farm were home-reared but there was no known contact between the infected child and the pigs. Three weeks after the onset of illness the boy was admitted to a London hospital. Details are given of the steps leading to a diagnosis of brucellosis. A blood culture taken on the 9th day after admission to hospital yielded an organism later identified as *Br. suis*.

Treatment with Aureomycin was followed by a complete disappearance of symptoms, and there had been no recurrence after three months.

Investigation of the pigs and cows on the farm by veterinary authorities have so far proved negative.

Chronic Brucellosis in General Practice. DAVIES, J. E. (1957). *Brit. med. J.* November 9th. 1,082–7.

This is the Charles Oliver Hawthorne Prize Essay for 1957 written by a general medical practitioner in Carmarthen. The investigation was carried out in general practice and was originally prompted by the fact that the writer himself contracted brucellosis 4 years ago. This personal experience led him to become aware that several patients, who were repeatedly attending his surgery on account of chronic and recurrent ill-health, had similar symptoms and signs to those he had himself experienced. The paper discusses 11 such cases, 2 of these had acute brucellosis in the beginning and after being followed up for nearly a year failed to show a return to normal health.

The general symptomatology of brucellosis in the human is described and details of the 11 cases are given in a Table. All cases had a positive skin reaction to brucellin, the incidence of dermal sensitivity to brucellin in a group of controls was found to be 21 per cent. The agglutination response to *Brucella abortus* in the patients of the series is given in detail in a Table; tests being continued at intervals for as much as 3 years in one patient.

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The relationship between brucellin injections and the agglutination response was studied and it was found that in a group of controls intradermal injection of brucellin resulted in the appearance of antibodies in a small number of cases. This non-specific rise in antibody titre, when it appeared, was neither of high titre nor sustained; the appearance of antibodies was not related to the original dermal sensitivity of the case.

Combined antibiotic treatment was used in all cases, the antibiotic used being tetracycline and streptomycin sulphate. Two cases responded to one course of antibiotic therapy, 5 responded to 2 courses, 1 to 3 courses, 1 to 4 courses and 2 which failed to respond to combined antibiotic therapy were given a second course combined with prednisone; they responded.

The possible disturbance of potassium metabolism resulting from the intracellular habitat of *Br. abortus* is discussed. It is pointed out that there has been "a considerable decline in the drinking of pasteurised milk largely as a result of publicity given to the Tuberculosis Milk Scheme." Tuberculin-tested milk has come to be regarded as a synonym for "safe milk" and little attention has been paid to the brucella risk.

Though it is not suggested that *Br. abortus* infection of cattle is more prevalent in the area involved than elsewhere it clearly is not insignificant from a public health point of view. Dalrymple-Champneys has estimated that 15 to 20 per cent. of adult cattle in Great Britain are infected. G. F. B.

In Parliament

Attested Herds (Irish Store Cattle)

MR. HURD (March 13th) asked the Minister of Agriculture, Fisheries and Food, in view of the progress being made with the attested herds scheme in Great Britain with the prospect that the whole country will be clear of bovine tuberculosis by 1961, what consultations he is having with the Irish authorities to ensure a supply of accredited Irish stock cattle that can be taken directly into attested herds in Great Britain.

MR. JOHN HARE: The authorities of the Irish Republic have throughout been kept informed of the progress of our attested herd scheme and they are well aware of the problems arising as the scheme approaches its final stage. Discussions are in progress with them about the future supply of store cattle, but I am not in a position to say more than this at the moment. I wish to assure my hon. Friend that I fully appreciate the importance of this matter.

MR. HURD: May we take it that my right hon. Friend is taking a personal interest in this problem, because he may well be faced with a position in which we may have an area clear of bovine tuberculosis here, and, although we still want Irish cattle, they cannot qualify to come in? Would not it be to the advantage of both countries if we could make mutually satisfactory arrangements as soon as possible?

MR. HARE: I can assure my hon. Friend of my concern in this matter, and I am giving it my very close personal attention.

Foot-and-Mouth Disease

MR. PEYTON (March 13th) asked the Minister of Agriculture, Fisheries and Food if he will enlist the co-operation of private enterprise concerns in foot-and-mouth disease research.

MR. JOHN HARE: Owing to the dangers of infection spreading from the handling of the virus and the unique facilities in the Research Institute at Pirbright, I am satisfied that Pirbright is the right place for research into this disease to be conducted. The Gowers Committee strongly supported this view.

MR. PEYTON: May I ask my right hon. Friend to have a look at this matter again? Is it not true to say that every effort, and as widespread an effort as possible, should be made to eliminate this scourge? Will he have some regard to the very considerable achievements made in similar fields to this by private industry? May I ask him if he will look at the matter again most carefully?

MR. HARE: Of course, I always listen to anything that my hon. Friend says, and I should like to extend to him an invitation to go to Pirbright himself to see the work carried out there. I should be delighted to arrange facilities for that.

COLONEL GLYN asked the Minister of Agriculture, Fisheries and Food how long the germ of foot-and-mouth disease can exist in the bone or bone marrow of infected South American cattle after they have recovered sufficiently to show no external symptom of the disease.

MR. GODBER: My right hon. Friend is advised that where cattle have recovered sufficiently from foot-and-mouth disease to show no external symptom, and have not been reinfected, the virus would not normally persist in the bone or bone marrow of their carcases.

COLONEL GLYN: I thank the Parliamentary Secretary for his Answer. Will he look further into the matter? Does he agree that there have been cases of infection in this country due to infected bones? While I accept the view of the experts that the virus does not generally persist, may I ask my hon. Friend to reconsider the issue?

MR. GODBER: We are always studying this very difficult problem. Its difficult feature is probably that one cannot rely on veterinary inspection to reveal that an animal is incubating the disease. That is probably the real cause of the trouble.

MR. ROYLE: Is not it possible for the disease to be carried even in boneless meat?

MR. GODBER: Yes, that is perfectly true. I am grateful to the hon. Member for bringing up that point. It is true that boneless meat can carry it, but such meat is not usually left about in the same way that bones are.

COLONEL GLYN asked the Minister of Agriculture, Fisheries and Food whether he will consider imposing regulations to prevent the spreading of foot-and-mouth disease carried by wrapping material from carcases imported from South America.

MR. GODBER: Such regulations are already in force. The Statutory Instruments are the Importation of Meat (Wrapping Materials) Order, 1932, as amended by the Order of 1939, and the Foot-and-

Mouth Disease (Packing Materials) Order, 1925, as amended by the Order of 1926.

COLONEL GLYN: I thank the Joint Parliamentary Secretary for that Answer, but is he aware that recent outbreaks have been traced to these wrappings, and that they have in fact been made use of in country districts in circumstances which can spread the disease? Is it possible to tighten up the regulations?

MR. GODBER: I am grateful to my hon. and gallant Friend for drawing attention to these regulations. It is our desire to see that they are effectively administered. If he can give me any information in relation to a particular case I shall be happy to follow it up.

Sheep (Foot-rot)

MR. CHAMPION: (March 13th) asked the Minister of Agriculture, Fisheries and Food what steps he has taken to bring the official leaflet, " Foot-rot in Sheep," to the notice of flock owners, with a view to the early eradication of this disease.

MR. GODBER: The leaflet is one of a series on animal health published for my Department by Her Majesty's Stationery Office. My veterinary officers bring the leaflets to the notice of individual farmers when appropriate and they are displayed at the Ministry's bookstalls at agricultural shows. We have also asked the National Farmers' Union to draw the attention of their members to the leaflets.

MR. CHAMPION asked the Minister of Agriculture, Fisheries and Food if he will make foot-rot in sheep a notifiable disease.

MR. GODBER: Measures to control this disease are well within the capacity of individual farmers, so my right hon. Friend does not propose to do this.

MR. CHAMPION: Is the Parliamentary Secretary aware that this is a painful disease which is costly to flock owners? Will the Government take every step possible to eradicate this disease which is eradicable?

MR. GODBER: I agree, and if I thought that it would help we would do what the hon. Gentleman asks, but I do not think it is necessary in a case like this. I think this is a matter where explaining to those concerned is the best way to deal with it.

Animals (Therapeutic Substances Act)

MR. LAGDEN (March 17th) asked the Minister of Health how many animals not covered by the restrictions of the Cruelty to Animals Act, 1876, were used during the year 1956, in premises licensed under the Therapeutic Substances Act, 1925, for the purpose of producing sera and related substances.

MR. WALKER-SMITH: About 480.

Sheep (Spraying)

SIR A. BOSSOM (March 17th) asked the Minister of Agriculture, Fisheries and Food if he will publish in the Official Report a statement explaining the further experimental work which has been done on the alternative method of using a spray instead of dipping sheep, to prevent sheep scab.

MR. JOHN HARE: Evidence has shown that thorough wetting of the skin is essential for the destruction of the parasites which cause sheep scab. Spraying is not a satisfactory alternative to dipping for this purpose because of the difficulty of treating unshorn sheep. No experiments have recently been undertaken in this country.

Milk Standards (Committee)

MR. GRANT-FERRIS asked the Minister of Agriculture, Fisheries and Food if he is aware of the fact that a leading official of the Milk Marketing Board has publicly advocated a lowering of the presumptive legal standard for solids other than fat in milk; and, in view of this, if he will make a statement of his policy in this regard.

MR. JOHN HARE: The presumptive legal standards for milk were enacted over 50 years ago and their suitability in present-day conditions has recently been questioned. My right hon. Friends the Home Secretary, the Secretary of State for Scotland, the Minister of Health and I have decided therefore to appoint a Committee to examine the problem both from the standpoint of human nutrition and of animal husbandry. It is hoped to announce the terms of reference and the membership of the Committee shortly.

MR. GRANT-FERRIS: While thanking my right hon. Friend for that Answer and assuring the House that the Question was in no way inspired, may I ask him if he would agree that the solid fat contents are as important as, if not more important than, butter fat, as they contain, amongst other things, those salts so important for the health of the general public, and children in particular?

MR. HARE: I think my hon. Friend will realise that that is exactly the sort of problem this Committee will have to study.

MR. CHAMPION: Is the Minister really satisfied that a further Committee is necessary? After all, was there not a Working Party on quality milk production which reported in 1953? Is it a fact that many of the recommendations of that Committee have not yet been carried out?

MR. HARE: I think the hon. Gentleman realises that there is a great deal of argument on all sides of this case, and I think that this Committee can do a useful job.

Dogs (Quarantine Regulations)

SIR R. ROBINSON (March 13th) asked the Minister of Agriculture, Fisheries and Food when the present period of quarantine on dogs entering the United Kingdom was first fixed; what is the present purpose of continuing this abnormally long period of quarantine; and whether in view of the constant improvement and greater security of modern preventive medicine, he will consider shortening this period.

MR. GODBER: The general requirement of six months' quarantine for dogs entering Great Britain from abroad dates from at least the beginning of the century, but with some variations before 1918. This quarantine period is necessary because the incubation period for rabies can last as long as six months and symptoms of the disease may not appear until towards the end of it. Rabies is a terrible and fatal disease and, like his predecessors, my right hon. Friend is not prepared to take the risk involved in shortening the period. Inoculation of dogs does not confer complete immunity.

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News and Comment

THE ROYAL SOCIETY OF HEALTH CONGRESS

The B.V.A. would be pleased to hear from members of the profession who hope to be present at this year's meeting of the Veterinary Hygiene Section. Names should be sent to the General Secretary, 7, Mansfield Street, Portland Place, London, W.1.

AGRICULTURAL RESEARCH COUNCIL VETERINARY RESEARCH FELLOWSHIPS

The Agricultural Research Council is offering two Veterinary Research Fellowships to provide opportunities for suitably qualified veterinarians to undertake research. The Fellowships are tenable for a maximum of 3 years and carry a stipend of £850 by £50 to £950 a year, plus superannuation. Candidates must be British subjects and hold a registrable veterinary qualification, and should have been engaged in research for not less than 3 years since qualifying.

The Council is also continuing to offer postgraduate research studentships to holders of veterinary qualifications, and a small number of veterinary studentships to enable honours graduates in one of the basic sciences to read for a veterinary qualification after taking their science degree. Applications for these awards should reach the Council by March 22nd.

Further details of the above awards, together with information about openings in agricultural research and advisory work, are obtainable from the Secretary, Agricultural Research Council, Cunard Building, 15, Regent Street, London, S.W.1.

THE IMPORTANCE OF *p*H IN THE MICROBIOLOGY OF MEAT

We are indebted to a correspondent for the following summary of a paper read before the Microbiology Group, Society of Clinical Industry, on December 5th, 1957, by Mr. M. Ingram, of the Low Temperature Research Station, Cambridge:—

It is well known that the pre-slaughter treatment of food animals has a decided influence on the glycogen content of the muscles and thus the consequent formation of lactic acid and the keeping properties of the food. Normally the *p*H of meat varies roughly between 5.5 and 6.5, and this paper discusses the microbiological effects of variations within this range.

Generally, spoilage bacteria are highly sensitive to changes in the acidity of their environment, and this is most important in the case of cured hams.

Acidity restricts the range of salt concentrations which the bacteria are able to tolerate, and has a directly inhibitory effect, particularly on those organisms which tolerate most salt. Furthermore, the inhibitory action of nitrite is greatest about *p*H 5.6. A relatively acid meat should also be advantageous in minimising the heat treatments used in the preparation of "pasteurised" as well as "sterilised" meat packs; because the heat resistance of vegetative cells, like that of spores, is diminished at a low *p*H.

The acidity also influences the colour and toughness of the meat, the exchange of water and the absorption of salt, the shrinkage on cooking, and the flavour when salted. In assessing the advantages of a low *p*H in the meat, this must be considered against a more salty flavour and greater cooking losses.

The preservative action of antibiotics is influenced by their stability at different *p*H levels. Weiser, Kunkle, and Deatherage found that chlortetracycline (Aureomycin) decomposed spontaneously in meat, reaching low levels (<1 p.p.m.) after a few days. The author suggests that the condition of the animal, acting through the *p*H of the meat, might be an important reason for such decomposition since chlortetracycline is much less stable at 7.0 than at *p*H 6.0.

THE COLONIAL VETERINARY SERVICE

Promotions. R. P. Lee, veterinary parasitologist, Federation of Nigeria, becomes officer-in-charge of the Animal Production Section of the Department of Veterinary Research, Federation of Nigeria; E. P. Lindley, S.V.R.O., Federation of Nigeria, becomes officer-in-charge of the Department of Veterinary Research, Federation of Nigeria; W. Plowright, senior scientific officer, East African Veterinary Research Organisation, Muguga, Kenya, becomes principal scientific officer, E.A.V.R.O., East Africa High Commission.

New appointment. R. A. Ayre-Smith, M.SC., DIP. AGRIC., Veterinary Officer, Kenya.

THE AYRSHIRE CATTLE HERD BOOK SOCIETY

At the Ayrshire Cattle Herd Book Society's annual conference at Eastbourne on March 6th, Dr. K. L. Blaxter of the Hannah Dairy Research Institute, Ayr, presented a paper, "The Nutrition of Dairy Cows." While Dr. Blaxter illustrated the influence of various levels of nutrition on production, he stressed the importance of good management. It was interesting that in the discussion, a member of the conference asked Dr. Blaxter if increased veterinary services were one of the expense factors which had to be remembered in high-producing herds. In reply Dr. Blaxter agreed that if one looked at the existing reports classified according to yield one did find veterinary attention at a higher figure in the high-producing herds. He contended, however, that adequate veterinary attention was simply a part of the good management which attended these high yields and that the farmer who calls in his veterinary surgeon for professional advice at an early stage is usually a far better manager than he who tries to economise in this direction, leaving the call until too late and finishing up with a dead cow.

FOOT-AND-MOUTH DISEASE

Foot-and-mouth disease was confirmed on March 14th among cattle at Southwick, Nr. Portsmouth, Hants. (within the existing Infected Area around Wickham, Nr. Fareham, Hants.).

The Infected Area has been extended to include all land within 5 miles' radius of the location of the new outbreak.

MR. PETER M. FLODIN

A. D. M. G. Laing and A. Mackay write from New Zealand:—

Peter Flodin's many friends were shocked to hear of his sudden death at the age of 40 just two days before Christmas, 1957. He died after only a few minutes' illness whilst on a farm in the Hutt Valley.

Born in Edinburgh in 1917 and educated at the Edinburgh Academy and the Royal (Dick) Veterinary College, Peter Flodin qualified in 1940. To his sorrow, the accidental loss of an eye just before his final examinations kept him out of World War II.

Peter Flodin came to New Zealand in 1943 after seeing practice as an assistant in Great Britain. After pioneering the formation of the Hauraki Plains Veterinary Club, Peter spent 8 years with the Opunake Veterinary Club. After a further 2½ years of practice with the Springs-Ellesmere Veterinary Club in Canterbury he joined the Animal Industry Division of the New Zealand Department of Agriculture in 1956.

The tributes to Peter Flodin which have come forward from all parts of the Dominion show very clearly in what high esteem he was held wherever he worked. In the untimely death of Peter Flodin the veterinary profession have suffered a real loss; Peter was conscientious, careful, and most thorough, and liked and respected by all with whom he came in contact, not least for the kindliness and consideration for others which were so characteristic of him.

Among Peter's interests outside the veterinary field his deep love of music was predominant, and he had acquired great skill as a pianist. He was also active on the committees of the Opunake and Leeston Dramatic Clubs and the Christchurch Recorder Society. At the time of his death he was a member of the Ohiro Bay School Committee, and of the Committee of the Ohiro Bay Parent-Teacher Association.

In 1942 Peter Flodin married Miss Adrienne Bernard, daughter of the late Mr. Bernard of the B.B.C. Symphony Orchestra, London. To her and to his two young daughters, Lesley and Jenny, we extend our most sincere sympathy.

CAPTAIN WILLIAM TEMPEST OLVER

Captain William Tempest Olver, who died at his home in Tamworth on February 1st, was a native of the borough, and was educated at the local Grammar School, Godolphin School in London, and Oundle, before proceeding to the Royal Veterinary College. He graduated with first-class honours in 1901, and went to South Africa with the Royal Army Veterinary Corps during the South African War.

In 1902 Captain Olver joined his father in practice in Tamworth. Two years later, on the death of his father, he took over the practice and the appointments of veterinary inspector for the counties of Stafford and Warwick. He received his commission in the R.A.V.C. in August, 1914, and was invalided out from France in September, 1918.

He returned to Tamworth and was elected to the Town Council in 1920, serving as a councillor and as an alderman until 1933. From 1929 to 1932 Captain Olver was mayor of the borough, and he became a Justice of the Peace in 1938. His death brings to an end a family record of public service to the district which stretches back over three generations—his father and grandfather before him also having served as mayors of the borough of Tamworth.

Captain Olver's first wife died in 1927, leaving a son and a daughter of the marriage; Mrs. Irene B. Olver was present at the funeral on February 5th. To his widow and to all his family we extend our deepest sympathies.

MR. BRIAN HAIGH

Mr. J. R. Holt writes:—

Mr. Sydney Jennings's appreciation of Brian Haigh in last week's VETERINARY RECORD prompts me to echo his praise of this charming person whom I regret I only came to know some two years ago.

His death is indeed of importance to us all for Brian Haigh was a hard and conscientious worker and we should take note of this lesson. He had his first attack of coronary thrombosis some time before I first met him.

His one ambition was to start practising again and he made several gallant attempts to do so, but each time was thwarted by a further thrombosis.

PERSONAL

Births

CAGIENARD.—On March 1st, 1958, to Anne, wife of B. Cagienard, B.V.M.S., 99, Ordsall Park Road, Retford, a daughter, Sally Anne.

HARBOURNE.—On March 16th, 1958, to Ann, wife of John Fulton Harbourne, B.V.SC., M.R.C.V.S., of 43, The Drive, Adel, Leeds, 16, a son, David William, brother for Elizabeth.

MACK.—On March 16th, 1958, to Doreen, wife of Roy Mack, M.R.C.V.S., of the Commonwealth Bureau of Animal Health, Weybridge, Surrey, a son, Peter, brother for Viki.

PHILLIPS.—On March 9th, 1958, to Myra, wife of George D. Phillips, B.SC., B.V.SC., M.R.C.V.S., of the East African Veterinary Research Organisation, Kikuyu, Kenya, a daughter.

STEELE-BODGER.—On March 13th to Anne (*née* Finlayson) and Alasdair Steele-Bodger, M.A., B.SC., M.R.C.V.S., of Pool House, Lichfield, Staffs, a sister for Catherine and Fiona.

Marriages

KING—BARRINGTON.—At Holy Trinity Church, Hermitage, on March 15th, 1958, John Stafford King, A.R.I.C.S., son of Mr. H. C. P. King, M.R.C.V.S., and Mrs. King of London, to Jennifer Ann, only daughter of Mr. and Mrs. E. Barrington of Hermitage, Berks.

SMITH—BRUNTON.—On March 15th, 1958, at Holy Trinity Church, Ashford, Bakewell, Derbyshire, Lawrance Blair, third son of George Blair Smith and the late Gertrude Blair Smith of Helensburgh, Dunbartonshire, to Mary Joyce, younger daughter of Dr. and Mrs. G. L. Brunton of Ashford.

COMING EVENTS

March

23rd (Sun.). First Meeting of the B.S.A.V.A. Western Region No. 7 at the Bristol Zoo, 2.30 p.m.

26th (Wed.). Annual General Meeting of the Scottish Metropolitan Division in the Royal (Dick) School of Veterinary Studies, 2.30 p.m.

27th (Thurs.). Annual General Meeting of the Western Counties Veterinary Society at the Rougemont Hotel, Exeter, 2.15 p.m.

Meeting of the Essex Veterinary Society at the Essex Institute of Agriculture, Writtle, Nr. Chelmsford, Essex, 7.30 p.m.

28th (Fri.). Annual Dinner-Dance of the East Midlands Division at the George Hotel, Nottingham, 7 p.m.

Meeting of the North Wales Division at the Council Chamber, Town Hall, Ruthin, 2 p.m.

Meeting of the Shropshire Veterinary Club at the Oak Hotel, Shrewsbury, 8 p.m.

April

2nd (Wed.). 20th Ordinary General Meeting of the A.V.T. & R.W.'s at Derby Hall, North Mossley Hill Road, Liverpool, 18, 10.25 a.m.

General Meeting of the Midland Counties Veterinary Society at the Feathers Hotel, Ledbury, 2.45 p.m.

9th (Wed.). Meeting of the Sussex Veterinary Society at the Old Ship Hotel, Brighton, 2.30 p.m.

11th (Fri.). Dinner and Dance of the West of Scotland Division in St. Enoch's Hotel, Glasgow, 7.30 p.m.

12th (Sat.). 12th Annual Dinner of the B.R.X. Club at the Royal Veterinary College, 6 p.m.

16th (Wed.). Combined Meeting of the Royal Society of Medicine, Section of Comparative Medicine, and the Section of Pathology at the Society's House, 5 p.m.

25th (Fri.). General Meeting of the Mid-West Veterinary Association at the Berkeley Café, Clifton, Bristol, 2.30 p.m.

May

10th and 11th (Sat. and Sun.). 6th Annual Reunion of the R.V.C. '52 Club at Ye Miller of Mansfield, Goring-on-Thames.

BRITISH VETERINARY ASSOCIATION QUARTERLY MEETINGS IN EDINBURGH

Wednesday, April 9th—Royal (Dick) School of Veterinary Studies

10.15 a.m. Small-Animals Committee.

2.15 p.m. Home Appointments Committee.

3.45 p.m. Farm Livestock Committee.

Thursday, April 10th—Royal (Dick) School of Veterinary Studies

9.30 a.m. Finance Sub-committee.

10.00 a.m. Veterinary State Medicine Committee. Committee.

11.30 a.m. Parliamentary and Public Relations Committee.

2.30 p.m. General Purposes and Finance Committee.

Friday, April 11th—Royal (Dick) School of Veterinary Studies

10.15 a.m. Council Meeting.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Aberdeen. Meikle Tarty, Ellon (Mar. 12).
Angus. Lunan Bank Farm, Arbroath (Mar. 14).
Wilts. Biss Farm, West Ashton Road, Trowbridge (Mar. 10).
Worcs Red Cross Farm, Perryfield Road, Bromsgrove (Mar. 10).

Foot-and-mouth Disease

Hants. Park Place Farm, Wickham, Fareham (Mar. 12); Lye Heath Farm, Southwick, Fareham (Mar. 14).

Fowl Pest

Lancs. Manor House Farm, Garstang, Preston (Mar. 10); 4, Kershaw Passage, Dearnley, Littleborough (Mar. 15).

Swine Fever

Beds. Brickwall Farm, Clophill (Mar. 10); Grange Farm, Cople (Mar. 13).
Bucks. Slough Bottom Cottages, Saunderton, Princes Risborough, Aylesbury (Mar. 10); Tythrop Park Farm, Kingsey, Aylesbury (Mar. 12); Hill Farm, Marlow Bottom, Marlow (Mar. 13).
Ches. Colshaw Hall Farm, Dean Row, Wilmslow; Oaklands Farm, Dean Row, Wilmslow (Mar. 14).
Hants. Cottagefold, Fishery Lane, Hayling Island (Mar. 14).
Leics. Nook Farm, The Nook, Great Glen (Mar. 12).
Norfolk. Bungalow Farm, Hoxne, Diss; 53, Ivy Road, Spixworth, Norwich (Mar. 13).
Northants. Hill View, Hollowell; St. James Allotments, Weedon Road, Duston (Mar. 10); New Cross, Gt. Brington; Piggeries next to Reading Room and Loose-box next to Public Baths, Gt. Brington (Mar. 13).
Salop. Sunnyside House, Coalbrookdale, Ironbridge (Mar. 13).
Warwicks. Middle Hill Farm, Wellesbourne (Mar. 10); Rudfyn Old Manor, Birmingham Road, Kenilworth (Mar. 11); Meres Farm, Pillerton, Heresey; Blackgraves Farm, Headley Heath, Kings Norton (Mar. 12).
Worcs. Park End Farm, Ribbesford, Bewdley; Ball Mill Top, Grimley (Mar. 12).
Yorks. Oak Butts, Towthorpe, Huntingdon (Mar. 11); Branch End Farm, Scholes, Cleckheaton; Field View, York Road, Haxby (Mar. 13).

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

The Diet of Sledge Dogs

Sir,—Britain maintains several prominent bases in the Antarctic, the largest being Hope Bay, 63° s., 57° w., where one of us (R.J.F.T.) spent the years of 1954 and 1955. At this time the complement of the base consisted of 12 men and about 75 Husky dogs. The diet of the dogs while at base consisted

of 6 to 8 lb. on alternate days of seal (usually *Lobodon carcinophagus*), most of which was muscle, fat, and bone, although a portion of heart and liver was incorporated. The practical experience of the previous 12 years indicated that this diet was adequate.

On journeys, however, a concentrated ration in almost dehydrated form must be provided, since on a loaded sledge about 50 per cent. of the weight carried may constitute food for the dog team. The ration has consisted since the time of Watkins in 1930 (see Watkins, 1932) of pemmican, the name given to a product based on beef. The approximate percentage composition of that in use during 1954-5 was beef meal 70, beef fat 22, dried brewer's yeast 4, bone meal 4. It was manufactured in solidified blocks, each weighing 1 lb., an average daily ration for one dog. In each 1 lb. there was an average of 0.40 mg. thiamine, 17.80 mg. nicotinic acid, and 1.46 mg. riboflavin, while vitamin A was not detectable. Fresh blocks contained rather less than 10 mg. per lb. ascorbic acid, and this had fallen to 3 mg. per lb. or lower after a year.

There had been complaints that pemmican alone was an inadequate sledging ration, and that it led to a deterioration in the condition of the dogs with loss of weight, almost constant diarrhoea, and, after about 6 weeks, the development of sore gums and palatal ulcers. It was recognised that dogs so fed became reinvigorated after being fed fresh seal.

Elsewhere, it was found that Huskies accompanying the British North Greenland Expedition became bored with pemmican, but would steal biltong, which they ate with avidity. These animals showed a marked tendency to eat human faeces (Masterton, Lewis & Widdowson, 1955; Lewis, Masterton & Ward, 1957).

Controlled observations at Hope Bay showed that dogs lost on average from 0.3 to 0.9 lb. daily, when receiving a daily ration of 1 lb. pemmican in addition to snow and water, the maximum loss recorded being 15 lb. in 9 days. Weight losses were found to occur even in non-working dogs. Limited observations indicated an increase in the urinary level of bisulphite-binding substances.

The pull of a team of 9 sledge dogs was measured electrically over a distance of 3,000 km., the daily individual ration being 1 lb. pemmican (Taylor, 1957), and it was computed that there was a decrease of work output of the order of 5 per cent. weekly.

While these observations were being made in the Antarctic, cognate studies in this country were conducted with a single Husky, and with other experimental dogs housed in metabolism cages and fed either pemmican or one of a series of diets of varying protein : fat : carbohydrate ratios. During the time that they received pemmican, the dogs showed changes comparable with those produced in the field, and these could be reversed by altering the dietary constituents so as to increase the proportion of carbohydrate.

These studies will be published in detail elsewhere, when acknowledgments will be made to the many persons who have assisted or advised at various stages. As a result of these investigations, it was possible to devise a diet which appears to be less wasteful or liable to set up deficiency conditions, and which was supplied to the Trans-Antarctic Expedition and for routine sledge journeys in a number of other countries.

Yours faithfully,
R. J. F. TAYLOR.

Department of Zoology, Cambridge, and
Falkland Islands Dependencies Survey.

ALASTAIR N. WORDEN.

Nutritional Research Unit,
Huntingdon.

C. E. WATERHOUSE.

Research Laboratories,
Bob Martin Ltd.,
Southport.

March 11th, 1958.

References

LEWIS, H. E., MASTERTON, J. P., & WARD, P. G. (1957). *Brit. J. Nutr.* **11.** 5.
MASTERTON, J. P., LEWIS, H. E., & WIDDOWSON, ELSIE M. (1955). *Proc. Nutr. Soc.* **14.** xvi.
TAYLOR, R. J. F. (1957). *J. Physiol.* **137.** 210.
WATKINS, H. G. (1932). *Geogr. J.* **79.** 466.

Transit Tetany

Sir,—Mr. Jarrett (*Vet. Rec.* **70.** 136) mentions transit tetany in cows. This responds rapidly to calcium by injection. I have been unable to find any published results of blood analysis in this condition, other than a suspected case recorded by C. Weighton (*Vet. Rec.* **54.** 49) which had serum levels of calcium 6.1, and magnesium 0.4 mg.p.c. I report two cases in the hope of hearing of further analyses.

Last December I attended two aged cows in transit by rail which had travelled some 160 miles. A Devon cow was laterally recumbent (heart rate 132, temperature 101.8° F.). Her muzzle was dry, and her pupil very dilated but sensitive to light. This cow was probably pregnant, but a rectal ulcer prevented confirmation by palpation. The other, a Guernsey cross, had a normal non-pregnant uterus. She was struggling to rise (heart rate 64, temperature 100.1° F.) and her muzzle was dry, although the pupil was normal.

Two hours after treatment with calcium borogluconate both cows were walking normally. The serum levels before treatment of the Devon cow were calcium 8.7 and magnesium 1.5 mg.p.c., and the Guernsey cross, calcium 9.0 and magnesium 1.6 mg.p.c. Although the blood magnesium was below the lower end of the normal range (1.8 mg.p.c.), it is not uncommon for cows to have a low blood magnesium level in the winter without showing any symptoms. The calcium levels were within the normal range.

Yours faithfully,
LIONEL A. HEMSLEY.

Clinical Department,
Royal Veterinary College,
Wallingford,
Berks.

March 14th, 1958.

Muscular Relaxants

Sir,—Mr. P. M. Keen gives three excellent reasons for condemning the use of succinylcholine and local anaesthesia in the horse.

Even if local anaesthesia were 100 per cent. certain in action, the psychological effect on the subject of induced total paralysis must always remain a humane objection to the use of this, and other curare-substitutes, on animals. Regarding the probable effect of this on the animal, and apart from the cogent evidence of subjective human experience, surely it is an accepted principle that anterior spinal anaesthesia is bad practice on account of the obvious distress that even paraplegia induces in animals—particularly large animals?

The fact that local anaesthesia even in expert hands is *not* 100 per cent. certain, while in the hands of the inexpert it is as likely as not to be unreliable, is surely a total bar to the use of the system under discussion. It is a clear-cut certainty, on the face of it, that an indeterminate number of patients *must* in the circumstances suffer operation without the benefit of anaesthesia. Indeed it would presumably be possible for any practitioner, using dud or defective anaesthetic, however competently, to go through the bottle and perform a number of operations, with no idea of the cruelty he would be perpetrating.

Dr. Hare suggests that succinylcholine (and like agents) should be scheduled as poisons, and otherwise treated in the manner of curare itself. It is a mystery to me how there can be any two opinions about this, or how the Statute Book can take such rigorous cognisance of the dangers of curare, while overlooking the very existence of its substitutes. If former legislators had good reason in the name of humanity to proscribe the use of curare on animals, why does present-day legislation fail so lamentably to cover the abuse of its synthetic equivalents? Or does this reflect that lower ethical and humane standards prevail to-day? Either the existing legislation affecting the use of curare is justified, or it is not. Does Dr. Montgomerie take the view that it is unnecessary and should be scrapped? Otherwise how can he advocate ignoring or scrapping exactly the same principles in respect of pharmacologically equivalent agents?

Dr. Montgomerie offers this method as a means of "avoiding much of the physical risk and mental torture associated with the accepted methods of casting the horse." I submit that so far as the factor "mental anguish" is concerned, the very reverse of what he suggests must be true.

Yours faithfully,

E. O. LONGLEY.

Abbotsford,
Goring-on-Thames,
Berks.

March 16th, 1958.

Treatment of Oedema Disease of Pigs with Vitamin B_1

Sir,—I was particularly interested in the clinical communication on vitamin B_1 by your correspondent, V. C. Ward of Warminster, on his successful treatment of "oedema disease." Some years ago, following a number of *post-mortem* examinations on the first pigs to die of this condition in this practice, I noticed the great similarity to the human condition, "beriberi." At a meeting of well-known representatives of fodder-merchants I spoke about this similarity and asked if they could discover whether, say, steam-hammer milling destroyed the vitamin B_1 during processing of the various grains composing their animal feeding-stuffs. I regret, however, that the authorities to whom I made my enquiries failed to reply!

Immodestly assuming that my suggestion hit the nail on the head and that future "bag-stuff" contained meal with its full quota of B_1—since "oedema disease" died out in this district some months later—perhaps the firm in question preferred not to admit that steam-hammer milling did in fact destroy this vitamin!

Mr. Ward's note has certainly rekindled my interest, and I now await the next "oedema disease" outbreak with real hopes of successful treatment. My advice to clients wanting to prevent this condition in the past was to add yeast to the feed twice weekly when they next heard of its incidence in the district.

Yours faithfully,

R. J. A. SWIFT.

12, Severn Street,
Welshpool,
Mont.

March 17th, 1958.

DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN
Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period		Anthrax	Atrophic Rhinitis	Foot-and-Mouth	Fowl Pest	Sheep Scab	Swine Fever
16th to 28th February, 1958		9	1	12	14	—	30
Corresponding period in	1957	21	—	—	29	—	30
	1956	212	1	4	13	—	25
	1955	33	—	—	29	—	53
1st January to 28th February, 1958		31	1	91	166	—	169
Corresponding period in	1957	72	2	11	191	—	139
	1956	500	2	4	125	—	103
	1955	184	—	5	140	—	193